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10/795,927	03/08/2004	Paul B. Fisher	A34694-PCT-USA-A 070050.2	2669
21003 7590 02/21/2007 BAKER & BOTTS L.L.P. 30 ROCKEFELLER PLAZA 44TH FLOOR NEW YORK, NY 10112-4498			EXAMINER WILSON, MICHAEL C	
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			1632	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	02/21/2007	PAPER

**Please find below and/or attached an Office communication concerning this application or proceeding.**

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

<b>Office Action Summary</b>	<b>Application No.</b> 10/795,927	<b>Applicant(s)</b> FISHER ET AL.	
	<b>Examiner</b> Michael C. Wilson	<b>Art Unit</b> 1632	

**-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --**  
**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

#### Status

- 1) ☒ Responsive to communication(s) filed on 13 November 2006.
- 2a) ☒ This action is **FINAL**.                      2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

#### Disposition of Claims

- 4) ☒ Claim(s) 61-69 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_\_ is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 61-69 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

#### Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

#### Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All    b) ☐ Some \* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
  2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
  3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- \* See the attached detailed Office action for a list of the certified copies not received.

#### Attachment(s)

- |  |   |
|--|---|
| 1) <input type="checkbox"/> Notice of References Cited (PTO-892)   | 4) <input type="checkbox"/> Interview Summary (PTO-413)<br>Paper No(s)/Mail Date. _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948)                       | 5) <input type="checkbox"/> Notice of Informal Patent Application                       |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08)<br>Paper No(s)/Mail Date _____ | 6) <input type="checkbox"/> Other: _____  |

### **DETAILED ACTION**

Applicant's arguments filed 11-13-06 have been fully considered but they are not persuasive.

Claims 1-60 and 70-75 have been canceled. Claims 61-69 remain pending.

### ***Specification***

The abstract of the disclosure has been entered.

The description of the drawings has been amended.

The title of the invention has been changed.

The nucleic acids in Fig. 1C have been labeled SEQ ID NO: 4 and 5 in the description of Fig. 1C.

This application repeats a substantial portion of prior Application No. 09/948227, filed 9-7-01, and adds and claims additional disclosure not presented in the prior application. Since this application names an inventor or inventors named in the prior application, it may constitute a continuation-in-part of the prior application. Should applicant desire to obtain the benefit of the filing date of the prior application, attention is directed to 35 U.S.C. 120 and 37 CFR 1.78. Claims 61-69 and 74 were filed with the instant application but were not present in parent application 09/948227. Section 5.6 of parent application has three paragraphs on pg 23, lines 11-30, while Section 5.6 the instant application has five paragraphs spanning from pg 22, line 19, through pg 24, line 2. Section 8 on pg 38-40 is not in parent application '227. The new paragraphs in Section 5.6 and new section 8.0 make the instant application a CIP of '227.

The first line of the specification will have to be updated to indicate parent case 09/948227 has been abandoned.

***Oath/Declaration***

The oath is correct.

Applicants confirm the instant application is a CIP of the PCT, which is a CIP of parent application 09/948,227 and state no revision of the specification or declaration should be necessary.

***Claim Rejections - 35 USC § 112***

***Enablement***

Claims 61-69 remain rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter that was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

**Breadth of claims**

Claim 61 is directed toward a transgenic mouse whose cells comprise the nucleic acid having the sequence of SEQ ID NO: 3 (human bivalent prostate carcinoma tumor antigen-1 (B-PCTA-1) protein; GenBank Accession No: L78132).

Claim 67 is drawn to a transgenic mouse whose cells express a greater level of PCTA-1 protein from expression of a the transgene, as compared to the level of PCTA-1 protein expressed in a non-transgenic mouse of the same inbred strain, and wherein the PCTA-1 protein has the sequence of SEQ ID NO: 6.

Claim 68 is drawn to a transgenic mouse having increased human PCTA-1 activity from expression of the transgene as compared to a non-transgenic mouse of the same inbred strain, and wherein the PCTA-1 protein has the sequence of SEQ ID NO: 6.

Claim 69 is drawn to a transgenic mouse whose cells express a greater level of PCTA-1 mRNA as compared to the level of PCTA-1 mRNA expressed in a non-transgenic mouse of the same inbred strain, and wherein the PCTA-1 protein encoded by the PCTA-1 mRNA has the sequence of SEQ ID NO: 6.

Claims 61 encompasses a transgenic mouse receiving gene therapy with a vector encoding SEQ ID NO: 3 as well as a transgenic mouse made using a transgene comprising SEQ ID NO: 3.

Claim 67 encompasses a transgenic mouse made using a transgene comprising any species of PCTA-1. Claim 67 encompasses a transgenic mouse receiving gene therapy with a vector encoding any species of PCTA-1 as well as a transgenic mouse made using a transgene comprising any species of PCTA-1.

Claim 68 encompasses a transgenic mouse expressing a transgene encoding human PCTA-1 as well as any transgenic mouse given human PCTA-1.

Claim 69 encompasses a transgenic mouse made using a transgene encoding any protein that causes increased levels of PCTA-1 mRNA, a transgene encoding any species of PCTA-1 that causes increased levels of PCTA-1 mRNA or any transgenic transgenic mouse given a protein that causes increased expression of PCTA-1 mRNA.

### **State of the art/unpredictability**

The state of the art at the time of filing was that the phenotype of transgenic animals was unpredictable. Examples in the literature aptly demonstrate that even closely related species carrying the same transgene construct can exhibit widely varying phenotypes. For example, several animal models of human diseases have relied on transgenic rats when the development of mouse models was not feasible. Mullins (1990, *Nature*, Vol. 344, pg 541-544) produced outbred Sprague-Dawley x WKY rats with hypertension caused by expression of a mouse *Ren-2* renin transgene. Hammer (1990, *Cell*, Vol. 63, pg 1099-1112) described spontaneous inflammatory disease in inbred Fischer and Lewis rats expressing human class I major histocompatibility allele HLA-B27 and human  $\beta_2$ -microglobulin transgenes. Both investigations were preceded by the failure to develop human disease-like symptoms in transgenic mice (Mullins, 1989, *EMBO*, Vol. 8, pg 4065-4072; Taurog, 1988, *J. Immunol.*, Vol. 141, pg 4020-4023) expressing the same transgenes that successfully caused the desired symptoms in transgenic rats. Thus, the phenotype resulting from a particular combination of elements (protein, promoter, species of protein, and species of transgenic) was not predictable at the time of filing.

B-PCTA-1 is part of the galectin gene family. B-PCTA-1 is referred to as bivalent because it comprises both carbohydrate recognition domains (CRDs). The specification teaches galectins are classified by distinguishing whether the galectin has a single CRD, two CRD domains separated by a linker, or an unrelated amino-terminal domain linked to a CRD (pg 2, 1<sup>st</sup> ¶ of specification). Galectins having tandem repeated CRD

domains include galectins 4, 6, 8 and 9 (pg 3, 1<sup>st</sup> ¶). Single CRD galectin 1 and 2 exist as dimmers (pg 3, 2<sup>nd</sup> ¶). It is based on the discovery that “increased expression of the full-length open reading frame of PCTA-1 suppressed proliferation of tumor cells while expression of PCTA-1 lacking the second CRD-encoding region had the opposite effect (pg 4, last ¶). B-PCTA-1 can be produced using a nucleic acid as set forth in GenBank Accession No: L78132, SEQ ID NO: 3 from residues 54-1004 (pg 12, 3<sup>rd</sup> ¶). The B-PCTA-1 protein is SEQ ID NO: 6, Fig. 10.

Gopalkrishnan (Oncogene, 2000, Vol. 19, pg 4405-4416) taught the precise biological functions for the galectin family as a whole, or for individual members has been elusive. PCTA-1 (closely related to rat and human galectin-8) was identified as a surface marker for prostate carcinoma. Overexpression of full length PCTA-1 inhibited growth of tumor cells *in vitro*. This observation is counter-intuitive to what one of skill would have expected of a potential oncogene (pg 4414, beginning of 2<sup>nd</sup> full ¶). The art at the time of filing did not teach the function of PCTA-1 *in vivo*.

#### **Teachings in the specification**

Increased expression of the full-length open reading frame of PCTA-1 suppressed proliferation of tumor cells while expression of PCTA-1 lacking the second CRD-encoding region had the opposite effect (pg 4, last ¶).

Applicants made transgenic mice made using a transgene encoding full length human PCTA-1 operably linked to the human elongation factor 1 $\alpha$  promoter (paragraph bridging pg 38-39). Applicants put the transgenic mice through a battery of tests to

determine the phenotype and found the transgenics did not have any observable phenotype (pg 39, lines 6-15).

Pg 40, lines 1-4, teaches transgenic mice overexpressing the protein encoded by SEQ ID NO: 3 crossed with TRAMP mice fail to produce detectable tumors as compared to TRAMP mice. Applicants conclude human PCTA-1 has a tumor suppressive effect.

### **Analysis**

While the specification teaches cells transfected with DNA encoding B-PCTA-1 may be used as a model of malignancy, transfection of cells with DNA encoding B-PCTA-1 inhibited tumor formation. Therefore the specification does not teach how to use the mouse claimed as models of malignancy.

Applicants made transgenic mice made using a transgene encoding full length human PCTA-1 operably linked to the human elongation factor 1 $\alpha$  promoter (paragraph bridging pg 38-39) and put the transgenic mice through a battery of tests to determine the phenotype and found the transgenics did not have any observable phenotype (pg 39, lines 6-15). Accordingly, it would require those of skill undue experimentation to determine the phenotype of the transgenic animal claimed. Without knowing the phenotype of the animal, one of skill would not be able to determine how to use the transgenic animal claimed. Therefore, the specification fails to teach how to use the transgenic mouse claimed because it fails to teach the phenotype of the transgenic mouse.

Pg 40, lines 1-4, teaches transgenic mice overexpressing the human PCTA-1 protein encoded by SEQ ID NO: 3 crossed with TRAMP mice fail to produce detectable tumors as compared to TRAMP mice. Applicants conclude human PCTA-1 has a tumor suppressive effect. Applicants do not teach how to use the PCTA-1/TRAMP mice for any further research. Nor are any uses for the singly transgenic PCTA-1 mouse readily apparent from such a conclusion. Furthermore, applicants are not claiming the doubly transgenic mouse. The teachings in example 8 do not provide adequate guidance for those of skill to use either the singly transgenic PCTA-1 mouse or the doubly transgenic PCTA-1/TRAMP mouse.

Applicants acknowledge that the normal physiological roles of galectins remain unknown (pg 3, line 1), it was unpredictable whether galectins were stimulatory or inhibitory because some galectins are stimulatory and some cause apoptosis (pg 37, 9-14). The specification does not teach the physiological role of B-PCTA-1 of a normal animal. In addition, different isoforms of B-PCTA-1 are expressed in different amount by a given cell population and possibly within the same cell at all times (pg 28, lines 15-30; pg 31, lines 3-5, "A complete lack of consistency PCTA-1 isoform expression has been observed in a given cell type"). The specification does not teach the function of B-PCTA-1 expression *in vivo* or the phenotype of any animal carrying a B-PCTA-1 transgene. The specification does not provide adequate guidance regarding the phenotype of the transgenic mouse claimed such that those of skill would overcome the unpredictability in the art and determine the phenotype of the transgenic claimed.

The specification does not enable one of skill to make a transgenic non-human animal whose cells comprise the nucleic acid sequence of SEQ ID NO: 3 as broadly claimed (claim 61). The claim encompasses administering the nucleic acid sequence of the transgenic mouse by gene therapy. However, the specification does not provide any means of providing gene therapy. Without such guidance, it would require one of skill undue experimentation to determine how to make a transgenic mouse whose cells comprise SEQ ID NO: 3 using gene therapy as broadly encompassed by the claim.

The specification does not enable one of skill to make a transgenic mouse whose cells express a greater level of any PCTA-1 as broadly claimed (claim 67). The only B-PCTA-1 transgene disclosed in the specification or in the art at the time of filing is the coding region of human PCTA-1 described in SEQ ID NO: 3 from residues 54-1004. The specification does not teach prostate carcinoma tumor antigens in any other species or how to isolate any PCTA-1 from any other species. It would require one of skill undue experimentation to determine whether other B-PCTA-1 exist or how to isolate such proteins. Therefore, one of skill could not cause greater expression of any PCTA-1 as broadly claimed.

The specification does not teach any transgenic having increased human PCTA-1 protein activity (claim 68) other than those having increased activity of the protein encoded by SEQ ID NO: 3. The specification does not teach how any other human PCTA-1 protein other than the one encoded by SEQ ID NO: 3. It would require one of skill undue experimentation to determine whether other human PCTA-1 proteins exist or

how to isolate such proteins. Therefore, one of skill could not cause greater expression of any human PCTA-1 as broadly claimed.

The specification does not teach how to increase human PCTA-1 protein activity (claim 68) other than by making a transgenic non-human animal whose genome comprises a transgene. The claim encompasses administering human PCTA-1 protein to any transgenic non-human animal and then finding a way to increase activity of the protein. No such teachings can be found in the specification and it would require one of skill undue experimentation to determine how to do so. Therefore, one of skill could not cause greater expression of human PCTA-1 in a transgenic non-human animal other than by making a transgenic non-human animal whose genome comprised a nucleic acid sequence encoding human PCTA-1.

The specification does not teach how to increase PCTA-1 mRNA (claim 69) other than by making a transgenic non-human animal whose genome comprises a transgene encoding PCTA-1. The claim encompasses administering a compound that stimulates PCTA-1 expression to any transgenic non-human animal. No such teachings can be found in the specification and it would require one of skill undue experimentation to determine how to do so. Therefore, one of skill could not cause greater PCTA-1 mRNA levels in a transgenic non-human animal other than by making a transgenic non-human animal whose genome comprised a nucleic acid sequence encoding PCTA-1.

Applicants argue in vitro studies showed PCTA-1 showed tumor suppressive activity (pg 32, lines 8-28; Fig. 8A-B) and that tumor suppressive activity would be expected in vivo. Applicants argue they have used PCTA-1/TRAMP mice to perform

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tumorigenesis studies. Applicants' arguments are not persuasive. Claims 61-63 and 67-69 are not limited to a PCTA-1/TRAMP mouse. Applicants have not provided a use for the singly transgenic PCTA-1 mice. Applicants have not shown PCTA-1 mice are resistant to tumors. Applicants have not taught how to use PCTA-1/TRAMP mice to study tumorigenesis or the function of PCTA-1 by teaching how to perform such "further research".

Applicants argue the PCTA-1 mouse can be used as a "research tool for specific purposes (enumerated below)." Applicants argue pg 23, lines 24-34; pg 24, lines 1-2 and pg 40, lines 1-4, describe the utility of the transgenic mouse claimed. Applicants' arguments are not persuasive. Pg 23, line, 26, through pg 24, line 2, states:

"The doubly transgenic B-PCTA-1/TRAMP animals also may be useful as models to determine the role of B-PCTA-1 on the processes of cell proliferation, cell migration and development. The apparent suppression of prostate tumor development observed in these animals suggests that B-PCTA-1 expression in vivo et situ can suppress tumor formation. Thus, further characterization of these animals may provide insights into the tumorigenic process. The animals may also be employed to identify agents that enhance the tumor suppressive effects, leading to the prostate. Insights gains from these studies may also be relevant to other tumor types in which autochthonous mouse models exist."

The specification does not teach how to use the PCTA-1/TRAMP mice to determine the role of PCTA-1 in cell proliferation, cell migration or development as suggested.

Therefore, the suggested use in the specification is so generic that it is meaningless.

Nor is it readily apparent that the PCTA-1/TRAMP mice will reveal the role of PCTA-1 in cell proliferation, cell migration or development. It would require those of skill undue experimentation to determine how to use the PCTA-1/TRAMP mouse to reveal the role of PCTA-1 in cell proliferation, cell migration or development. Pg 40, lines 1-4, merely

teach that PCTA-1/TRAMP mice were free of detectable tumors without teach how to use the mice for further research.

Pg 22, lines 19-31, relates to using transformed cells in vitro and not the transgenic mouse claimed.

Pg 23, line 30, through pg 24, line 2, are discussed above and fail to teach the assays required to use PCTA-1/TRAMP mice to determine the role of PCTA-1 in cell proliferation, cell migration or development. Assuming such assays are readily apparent to those of skill in the art, it is not readily apparent that any assays will determine the role of PCTA-1 in cell proliferation, cell migration or development.

Applicants argue the mice of claims 64-65 are a doubly transgenic mouse equivalent to a PCTA-1/TRAMP as specified by claim 66. Applicants' argument is not persuasive. The mice of claims 61-65 and 67-69 do not relate to TRAMP. Only claim 66 has the same structural features as TRAMP mice, i.e. a construct encoding SV40 T antigen operably linked to the rat probasin promoter. Claims 64 and 65 require the mouse comprises a nucleic acid sequence encoding SV40 T antigen, but it is not expressed specifically in prostate or linked to a prostate specific promoter. The specification does not provide adequate guidance for those of skill to use the PCTA-1/TRAMP mice to determine the role of PCTA-1 in cell proliferation, cell migration or development; therefore, the specification does not enable using PCTA-1 the transgenic mouse claimed.

### ***Indefiniteness***

The following is a quotation of the second paragraph of 35 U.S.C. 112:

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The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 67-69 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 67-69 are indefinite because "the transgene" lacks antecedent basis. While the claims are directed toward a transgenic mouse, the claim does not clearly set forth the mouse has a transgene. The phrase "transgenic mouse comprising a transgene..." would overcome this rejection.

Claim 69 as amended is indefinite because it does not clearly set forth the PCTA-1 mRNA encodes the amino acid sequence of SEQ ID NO: 6. "The protein" lacks antecedent basis and the phrase added is confusing. Clarification is required.

***Claim Rejections - 35 USC § 103***

Claims 61, 62, 67-69, 74 and 75 remain rejected under 35 U.S.C. 103(a) as being unpatentable over Kasper (1998, Laboratory Invest., Vol. 78, No. 3, pg 319-333) in view of Su (PNAS, July 1996, Vol. 93, pg 7252-7257) for reasons of record.

Kasper taught a transgenic mouse whose genome comprised a transgene comprising rat prostate tumor-specific antigen (probasin) operably linked to a promoter. The transgenic mouse overexpressed probasin; therefore, the mouse had an increased probasin activity and mRNA levels. Kasper did not teach making a mouse whose genome comprised human PCTA-1.

However, Su taught the nucleic acid sequence of SEQ ID NO: 3 (GenBank No: L78132) for human prostate carcinoma tumor antigen-1 (PCTA-1) (pg 7252, col. 2, "Data Deposition" at the bottom of the page), which encodes SEQ ID NO: 6.

Thus, it would have been obvious to one of ordinary skill in the art at the time the invention was made to make a transgenic mouse whose genome comprised a prostate tumor antigen from a non-mouse species as taught by Kasper, wherein the prostate tumor antigen was the human prostate carcinoma antigen encoded by SEQ ID NO: 3 taught by Su. One of ordinary skill in the art at the time of filing would have been motivated to replace the rat prostate tumor antigen used by Kasper with the human prostate tumor antigen taught by Su to determine the function of PCTA-1 *in vivo*.

The specification teaches transgenic mice comprising SEQ ID NO: 3 did not have altered phenotypes (pg 39, lines 6-18). Therefore, the combined teachings of Kasper and Su are no less than the teachings in the specification.

Applicants argue "there would be no rational basis to combine the disclosures of Kasper and Su due to the fundamentally distinct nature of the combination compared with the presently claimed invention. First a mouse based on Kasper would only have restricted prostate specific expression. An important basis of the present invention is to be able to study several tumor types... ..which preferably requires ubiquitous PCTA-I transgene expression." Applicants' argument is not persuasive. Applicants used and claim using the human elongation factor 1 $\alpha$  promoter (claim 63), which does not provide ubiquitous transgene expression. Claim 63 is the only claim limited to the human

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elongation factor 1 $\alpha$  promoter but is not rejected under 103 using Kasper and Sun. The rejected claims do not require ubiquitous transgene expression.

Applicants argue the phenotype of the single transgenic mouse taught by Kasper is tumorigenic not tumor-suppressive. Applicants' argument is not persuasive because the claims do not require a phenotype.

### ***Conclusion***

The prior art made of record and not relied upon is considered pertinent to applicant's disclosure:

HomoloGene shows PCTA-1 in humans, mice and rats is now known as LGALS8 (lectin, galactoside-binding, soluble, 8 or galectin 8).

No claim is allowed.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire **THREE MONTHS** from the mailing date of this action. In the event a first reply is filed within **TWO MONTHS** of the mailing date of this final action and the advisory action is not mailed until after the end of the **THREE-MONTH** shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of

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the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Inquiry concerning this communication or earlier communications from the examiner should be directed to Michael C. Wilson who can normally be reached at the office on Monday, Tuesday, Thursday and Friday from 9:30 am to 6:00 pm at 571-272-0738.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

Patent applicants with problems or questions regarding electronic images that can be viewed in the Patent Application Information Retrieval system (PAIR) can now contact the USPTO's Patent Electronic Business Center (Patent EBC) for assistance. Representatives are available to answer your questions daily from 6 am to midnight (EST). The toll free number is (866) 217-9197. When calling please have your application serial or patent number, the type of document you are having an image problem with, the number of pages and the specific nature of the problem. The Patent Electronic Business Center will notify applicants of the resolution of the problem within 5-7 business days. Applicants can also check PAIR to confirm that the problem has been corrected. The USPTO's Patent Electronic Business Center is a complete service center supporting all patent business on the Internet. The USPTO's PAIR system provides Internet-based access to patent application status and history information. It also enables applicants to view the scanned images of their own application file folder(s) as well as general patent information available to the public.

For all other customer support, please call the USPTO Call Center (UCC) at 800-786-9199.

If attempts to reach the examiner are unsuccessful, the examiner's supervisor, Peter Paras, can be reached on 571-272-4517.

The official fax number for this Group is (571) 273-8300.

Michael C. Wilson

A handwritten signature in black ink, consisting of a series of loops and a long horizontal stroke at the end.

**MICHAEL WILSON  
PRIMARY EXAMINER**